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The venous pulse

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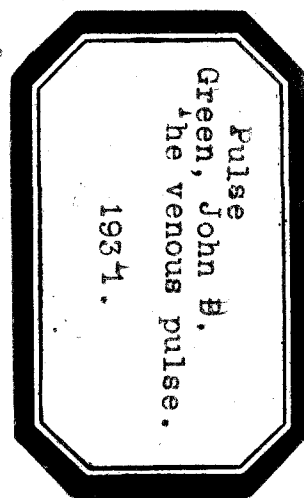
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- THE VENOUS PULSE -

by

JOHN D. GREEN

A Senior Thesis

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INTRODUCTION

Taking as a major premise that a thorough understanding of the mechanism of production and time relations of the various waves of the venous pulse is fundamental to a proper interpretation of the individual record, the purpose of this paper is to attempt a brief but representative survey of the findings of previous investigators and to correlate these findings with the writer's investigations. This will be undertaken by a brief discussion of the following subjects:

Early history.

Nomenclature.

Interpretation of the various waves of the venous pulse.

Writers' investigations and observations.

Clinical applications.

Conclusions.

EARLY HISTORY

Rhythmic pulsations of the large veins of the neck were reported as early as 1704 in a case of cardiac dilatation by Homberge. No great progress as to the interpretation of the pulsations occurred until after the invention of the sphygmograph by Marey in 1860 (16). He published the first graphic records in 1863 and concluded that the venous pulse was a sign of tricuspid incompetence. Friedreich published venous curves from the neck of patients in 1866 (15). A great forward step were the records of Potain who was first to publish simultaneous carotid and venous pulse.

There has been no essential alteration of the fundamental principle for registration of the pulse since those early days. But as the sensitivity of recording tambours increased with the development of Franks segment capsule (12) and its modifications (29-46-64-65-66), more details of the venous pulse have been revealed (49-51-56-58-61).

Mackenzie early suggested that pulsations in the vein bear a relation to the right heart comparable to that which the arterial pulse bears to the left (36). More recent investigators have shown the similarity to the pressure curves taken directly from the auricle to the venous pulse; and it has become increasingly obvious that a proper understanding of the venous pulse may be of

service in the recognition of the successive normal and pathological dynamic events in the heart, in deciphering the mechanism of the arrhythmias, and in giving clues to clinical signs.

At least two definite forms of the venous pulse have been recognized since the earliest publication of graphic venous pulse records. They have been classified by Mackenzie (36) as the "auricular venous pulse" and the "ventricular venous pulse". He decries the use of the terms negative or normal for the former and positive or pathological for the latter, as is often used (39). Other terms which have been applied to the negative or normal pulse are physiological venous pulse (44), systolic venous collapse (1), double (27), and false venous pulse (36). The positive or pathological pulse has also been called the centrifugal (42), regurgitant (1) or true venous pulse (24-36). White, who has described a centripetal venous pulse, says a pulse may pass from the arterial to the venous side of the circulatory bed in normal subjects (60). Such a pulse has also been described in pathological subjects (5-41).

The negative quality of the venous pulse was recognized as early as 1796 by John Hunter (28). He thought it was "probable that -- the action of the arteries and veins is alternate". He observed that "when the arteries contract -- the veins dilate". Mosso was probably the first to record that the pulsation in the vein was

essentially a negative pulse, inasmuch as there was a great fall during the ventricular systole.

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NOMENCLATURE

The waves of the venous pulse have been variously termed by investigators according to the different interpretations of their causes.

As many characteristic venous pulse records have been recorded as there are investigators (1-3-7-11-21-22-26-32-36-37-40-44-46-49-51-56-58-61-65). The differences being due to differences in the sphygmograph, light or firm pressure on the vein, central or lateral location of the receiving tambour with respect to the vein, frequency of recording tambour, presence of impact waves, absence or character of respiration, presence of pathology in the heart, venous pressure or engorgement, choice of vein, and mechanical understanding of the operation. Fig. 1 shows a characteristic phlebogram as recorded by the writer.

The nomenclature used by Lewis (34) is as generally accepted as any. He calls the three waves A, C and V, the waves being approximately presystolic, systolic and diastolic in time. The same terminology is used by Mackenzie who first used it. He speaks of the first as the A wave or auricular wave, the second as C or carotid, and the third as V because of "its association with systole of the right ventricle" (38). He designates the systolic fall as X and X', and the diastolic fall as Y.

The A wave has been designated as the "P" wave as it is presystolic in time (2). Between the A and C waves there has often been described a second A wave (9) or intrasystolic wave (1-2-9-27-64). Hering has called it the Ad or auricular diastolic wave (23).

The C wave is often called the systolic or S wave (1-14-45-46). Morrow has also called it the ventricular wave (44). He dislikes the term C. The German investigators often designate it as the Vk (7-22) or K wave (23).

Morrow suggests that the V wave be called the "first onflow wave" normally and the back flow wave in tricuspid regurgitation (45). Porter calls it the diastolic wave (47). Hirschfelder has called it the stagnation wave (25). Many investigators have noted that the upstroke of the V wave is divided by a notch or bend and different names have been attached to the two parts. Bard terms them T and D, that is telosystolic and proto-diastolic (2). Ewing designates the first part as the first onflow O, or pre-diastolic and the second portion as D or diastolic (9). Edens terms them Vs due to stases and Vd due to displacement of the base of the heart.

Many have described or published records of a notch or series of vibrations on the upstroke of the V wave (1-2-3-11-24-27-35-39-40-46-51-54-55-56-61-65) which I have designated as the incisura of the V wave or iV. Some work carried out by the writer in the physiological

laboratory of the University of Nebraska College of Medicine may serve to shed some light as to the time relations of this incisura.

Other waves occurring between the V and A waves have been designated as the H wave (26), B wave (18), second onflow wave (9-45), stasis or S wave (24), X wave (10), W wave (27), second diastolic wave (68), and sinus wave (20).

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THE INTERPRETATION OF THE VARIOUS WAVES OF THE VENOUS PULSE

The A Wave.

The rise of the presystolic or A wave is almost invariably interpreted as being due to the contraction of the auricle (1-9-21-27-34-35-39-55-61-64). The veins swell because the entry of blood to the auricles is opposed by auricular contraction (35). There is a sudden increase in the diameter of the veins either the result of damming of the blood, or a wave of regurgitation as suggested by Bachmann (1). Keith presents morphological evidence that a band of muscle fibers, the tinea terminales, serves to close off the large venous ostia during auricular contraction (30). However, Straub (51) and Wiggers (62-64) and others (54) have demonstrated definitely that the A wave though belated, rises and falls synchronously with the intra-auricular pressure tracings.

Morrow (43) has determined the jugular transmission time to be 1-3 meters per second which accounts for the lag.

The Systolic Wave.

The second positive wave - the S or C wave - has been much discussed. Mackenzie regarded its rise as caused by, and hence synchronous with carotid impact (36-37-38-39-40). He goes as far as to say that it is absent if not transmitted by the carotid (38). Other able investigators support him in this position (17-59). He with other relatively modern investigators believed that the "true venous pulse" has no C wave (24-37).

Bard, however, found that the C wave preceded the carotid wave by .01 to .02 seconds (2). This fact was given greater significance when Morrow demonstrated a pulse wave velocity in the jugular vein equal to about one-half that in the arteries (43). Many other investigators agree that the systolic wave of the venous pulse precedes the arterial rise (1-4-8-9-11-14-26-27-44-46-56).

Morrow, moreover, demonstrated the C wave in the isolated jugular of dogs where there was no possibility of arterial impact (45). He suggests that factors in the production may be: a force exerted during ventricular systole through the a-v valves; a contraction of the ring of muscle around the a-v junction; the result of pressure exerted upon the auricles by a systolic twist of the heart (44).

Many investigators subscribe to the idea that the beginning of the C wave may be caused by the closure or

bulging of the auriculo-ventricular valves (2-14-21-27-34-35-44-46-47-61). Edens recorded optic venous tracings in which the C wave had a small primary rise which he interpreted as being due to closure of the a-v valves, and a subsequent sharp rise due to arterial impact (7).

The presence of a systolic wave in intra-auricular pressure curves (47-51-54-62-65) lends opposition to the pure arterial impact theory of Mackenzie (36-37-38-39). But there is no doubt that in most venous tracings as recorded from the jugular vein, there is some arterial element. The question as to whether the S wave from the auricle or the arterial impact predominates, and the relation of each appears to be the chief points of disagreement.

Very good observers have changed their opinion on the subject. Mackenzie in 1907 would accept none but the pure arterial hypothesis. But in his last publication in 1925 he mentions "faint waves in the jugular pulse that may occur" - "about the time of the carotid wave; produced in some 'obscure way' by the 'systole of the ventricle'" (40).

Wiggers in 1915 speaks of a "systolic wave" in the jugular which "precedes the rise of the subclavian very definitely". He mentions "a notch corresponding to a similar rise in the auricle" which is "cut short by a prominent elevation C synchronous with subclavian rise or arterial impact". He regarded the "introductory rise" as

"due to closing the tricuspid" and as resultant of the sharp vibration recorded from the auricle at the beginning of systole (61). In 1923 he summarizes his views as follows: "There exists in the right auricle a small systolic wave due to the movements of valves or the a-v floor; there exists in the central veins (occasionally in the auricle) another distinct wave due to an intra-thoracic impact; the latter is and the former is not transmitted to the veins of the neck; it is difficult to record either of these systolic vibrations when a small tambour simultaneously covers an arterial area, for then the period of systole is obscured by a direct arterial impact (64).

Another element which is said to be a factor in the production of the wave is a sudden ejection of blood from the coronary sinus (27-47).

Lewis concludes that it cannot be affirmed at present that the C wave is purely arterial or purely venous in origin, and while it cannot be denied that both factors may be contributory under certain conditions, it is highly probable that in one case the arterial and in another the venous element will predominate (34).

Whatever the cause of the C wave, it is very valuable clinically indicating as it does the onset of ventricular systole.

The Systolic Collapse.

According to Mackenzie (40) the fall of the A wave is due to three factors, i.e. the relaxation of the

auricle after its systole; the dragging down of the a-v septum by the ventricular muscle enlarging the auricular cavity; and the diminished intra-thoracic pressure in consequence of the expulsion from the chest of the contents of the left ventricle. The first element mentioned is most often given as the cause, that is the collapse of the veins occurring with resumption of blood flow consequent on relaxation of the auricle (1-21-55).

Ewing (9) points out that the presystolic fall takes place during auricular systole and inclines to the theory of a rebound of blood following an impact wave caused by auricular systole. Wiggers agrees that time relations make it necessary to explain the pressure decline during continued auricular systole. He interprets the fall as due to the "progressive elimination of fractionate contractions from the auricular contraction process" (64).

The fall after the C wave, according to Mackenzie, is merely the continuation of the decline after the A wave and is due to the same factors (40).

Lewis, after summarizing the current theories, concludes that the two negative waves which together form the most prominent depression of the auricular and venous curves are due to three causes; the increased negative pressure in the chest consequent on ventricular systole; the auricular relaxation dependent on the original intra-pleural pressure; and the stretching of the auricular walls

as a result of ventricular systole. "Of these causes, the first is insignificant; the second is most active during the early phases and the third most prominent in the later phases of the depression" (34).

Other factors which have been represented as having a major part in the production of this depression are, the traction on the vena cava and great vessels which increase their capacity and pumps their contents into the auricles (27-51); and the contraction of the papillary muscles (1-47).

The V Wave. (Onset)

There is a great deal of variation in the reported time relations of the beginning of the V wave. Wenckebach believed that it began synchronously with the second aortic sound (59). Ewing published records showing it begins at a fixed point early in ventricular systole viz., at the end of auricular relaxation (9). Gerhardt times its occurrence with the commencement of the diastolic wave (17). Mackenzie found that the beginning of the rise was very variable and dependent on the amount of blood in the auricle and large veins, and the degree of tricuspid regurgitation (36-37). Eyster found that it could begin either before or after the second sound but that on an average the rise was a few hundredths of a second before the second sound (11). Veiel and Kapff believed the beginning of the carotid incisura and the rise of the V wave to be from the same cause; the difference being due

to transmission time (56).

Straub published records showing that the wave usually began very late in ventricular systole but believed its rise depended on the amount of blood in the venous reservoir; the flow from the periphery; and the traction on the auricles and veins (51). Wiggers (6), Weber (56), and Edens (7) published optic records that show the third wave as essentially and sometimes entirely diastolic in time, i.e., after the closure of the semilunars. Wiggers believes that the foot of the V wave is either late in systole or early in diastole, depending on the declining gradient of the impact wave (65).

Edens after summarizing the various opinions as to the time of the beginning of the V wave concludes that it varies in different individuals, as well as in the same individual (7).

Mackenzie in his earlier publications (36-37-39) inferred that tricuspid regurgitation was a normal physiological event, and was a constant factor in the production of the V wave. Gibson (19) and other early investigators inclined to the same opinion. Mackenzie explained the delay in the appearance of the V wave after ventricular systole by the fact that there interposed a dilating auricle and a capacious superior vena cava between the ventricle and jugular bulb. He worked out a schematic diagram which illustrated how the V wave would appear progressively earlier with increasing tricuspid insufficiency (36-37).

Vaquez states that the rise of the V wave corresponds to the end of the emptying of the ventricle and consequently to the filling of the auricle, which produces the swelling of the jugular system and the elevation that represents it. This filling of the auricle may be more or less premature according to whether or not there exists a stasis of blood in it (55).

The V Wave. (Interpretation)

The variations in the interpretation of the V wave correspond to the differences of opinion in regard to the instant of its onset. The explanations obviously depend upon the time relations that the particular investigator accepts. Those who believe that the onset is during the systole of the ventricle incline toward the stasis theory (22-39-44-47-51). While those who regard it as rising with the beginning of ventricular diastole attribute it to some diastolic event occurring during the isometric relaxation phase (3-7-14-17-47-59). Many accept a compromise view, i.e. that it is caused by combination of factors (1-2-3-7-9-27-34-40-46-47).

It has been observed that the V wave is exactly like the A or C wave in type. The stasis theory of its production has been questioned on this account, and some more rapid acting force suggested as the cause (50). Barry agrees that the sharp apex of the V wave must be accounted for by some more rapidly acting force than the simple ending of a period of stasis. He suggests that two things happen

about the moment that the summit of the V wave is to be formed, i.e. the swing of the semilunar valves and the increase in the intra thoracic pressure at the beginning of diastole. He believes that either one or both of these events may act to change what would otherwise be a slowly formed shoulder to a sharp peak (3).

Riegel believed that a factor in the production of this wave was the dicrotic rebound transmitted from the arteries to the vein (48). Francois-Franck ascribed it to a wave transmitted back through the ventricle and auricle at the moment of closure of the pulmonary valves.

Grosh and Cushney present what they believe to be unanswerable evidence against stasis being the chief cause of the V wave. They published records which show that the V wave is dependent upon ventricular systole (21). Morrow's conclusion from his records were just the opposite. He was convinced that the action of the ventricle is quite unnecessary for the production of the V wave and believed it is produced as Gottwalt suggested in 1851, simply from the onflow of blood from the systemic arteries and capillaries which refills the veins just emptied by auricular diastole (44).

Ewing divided the ascending limb into two parts. The first due to rapid onflow and the second continuous with the upward movement of the base of the ventricle with beginning relaxation of the ventricle. He states that a

small onflow wave may be present, but that Vd is absent when the auricle beats and the ventricle does not. He suggests that the V wave may be the superimposed combination of the two factors due to the fact that the stasis wave travels slower and reaches the neck simultaneously with the second wave (9).

Bachmann believed that the two lesser waves in the V wave were due to the relaxation of the papillary muscles at a time when the intraventricular is still higher than the intraauricular pressure, this resulting in an upward movement of the tricuspid leaflets, and to a return of the a-v septum to its position of rest (1). Bard described the two portions as coincident with the pre-dirotic wave and with the vibration of the ventricle due to closure of the semilunars (2).

It does seem logical that if the descent of the a-v line is sufficient to produce the negative wave (X-X'), that its rise should be adequate to produce a second wave of equal intensity and opposite sign. The only difficulty according to Lewis (34) is that this wave if propagated may be entirely swamped by other events occurring at or about the same time.

In 1909 Lewis believed that two factors, namely, stasis, and the upward spring of the a-v junction at the beginning of diastole played a part in the production of the V wave under certain circumstances. The first factor

he thought to be prominent with a quick filling of the auricle or sustained plateau. But with slower filling and quicker heart beat the pressure in the auricle will be low when the ventricle passes into diastole and as a consequence the tricuspid valves will open later. Under these circumstances the second factor may be more pronounced. He advanced this view to promote harmony and to account for the division of the wave at or about the time of closure of the semilunars (34). However, in 1924 he states that the V wave in veins is normally a pure affair of stasis (35).

Wiggers is tactfully noncommittal as to the cause of the V wave (65). He believes if mechanical factors contribute - outside of stasis - it can only be said that their nature has not been clearly defined. (64).

The Incisura of the V Wave.

As was indicated under the discussion of nomenclature, a large number of investigators have described or published records showing a notch or series of vibrations at the foot or on the ascending limb of the V wave. Some authors are noncommittal and others very positive as to the cause of these vibrations. Those who divide the V wave into two separate waves with distinctly different causes, believe that the notch merely represents the end of the first or the beginning of the second force (1-2).

Others have described it as synchronous with the second sound (11-35-51-56-65), the dicrotic notch (56),

the end of systole (27-65), pulmonary closure (40-43-56), a similar notch in intra auricular pressure curves (54-61), or of obscure origin (39).

Barry has often noted in triple records - jugular, carotid, heart sounds - a series of vibrations corresponding to the second sound, on the dirotic wave as well as on the upstroke of the V wave, with exactly the same frequency. He believes that the vibrations reach the vein through auricular conduction as he finds in many curves taken from the right auricle or superior vena cava in dogs a series of vibrations corresponding in time and rate to the second sound (3).

The Fall of the V Wave.

The fall of the V wave "Y" is universally attributed to the opening of the a-v valves (1-3-27-32-35-39-40-44-51-54-55-61-64-65). Mackenzie states that the termination of the V wave is "one of the most certain periods in the cardiac cycle indicating as it does the opening of the tricuspid valves" (40).

Practically every author sometime or other during his discussion of the venous pulse observes that the drop of the V wave occurs in both auricular and venous curves synchronous with the opening of the a-v's or they refer to the unanimous agreement on the subject.

Lewis raised his head above the tide long enough to state that "the interpretation of the 'Y' depression may be correct, but it has not been arrived at by scrupulously

accurate methods" (34). This is a very accurate even though a trite statement.

The Diastolic Waves.

The second diastolic wave is not constantly present but it nearly always occurs in long cycles. Hirschfelder (26) and Gibson (18) separately described it and attributed it to a sudden floating together of the a-v valves at the end of the period of rapid inflow. They believed it to be associated with the third sound and were confirmed in this by Thayer (53) and Eyster (11).

Morrow (44) previously had described a second onflow wave which subsequent investigators (9) took to be the same as the Hirschfelder's wave.

Another wave has been described which occurs in late diastole (9-10-20-27), the W (27), X (10) or sinus wave (20). It has been attributed to a rhythmical contraction of the muscular fibers of the superior vena cava (20) and to the contractions of embryonic tissue at the mouth of the vena cava (9).

Eyster states that this X wave is not as frequently present as is the H wave and believes that no wave can be referred to the contraction of the region of the great veins or sinus region of the heart (10).

Variations in the Interpretation of the Waves.

The differences in the interpretations of the venous pulse have apparently several reasons. There is

undoubtedly considerable variation in the form of the pulse for reasons listed previously in this paper.

Writers have a tendency to refer certain waves to standard events with insufficient or at least unpublished evidence; most of the views advanced being but plausible suggestions compatible with the time relations of the waves. A later investigator, if he happened to use an erroneous conclusion, is thus led into further error. Writers often report their findings in such a way that it is difficult to be certain whether they are referring to the actual time relation of an event or to a curve in a tracing which is supposed by them to be synchronous with that event.

Modern investigators have a tendency to discredit all previous findings that do not correspond to their own ideas, using the fact that the lever inertia or the frequency of the apparatus used might complicate the curves it produced.

Other sources of error have been synchronous points; parallax in optical methods; differences in arterial and venous pulse wave transmission time relatively and during different parts of the cardiac cycle; tube transmission time; and relative location of receiving tambours. In their attempts to evaluate these factors some have gone further astray than their predecessors and have published corrected records which are dynamically impossible.

It is therefore not surprising that different investigators should arrive at different interpretations of synchronous events. Wiggers states that all corrections must be tempered with a consideration as to whether the dynamic events thus indicated are possible. He has many tracings in his possession which, if he "adhered to strict corrective processes, would force one to admit that a condition of dynamic chaos must exist in the cardio vascular mechanism" (63).

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WRITER'S INVESTIGATIONS AND OBSERVATIONS

The writer's interest in an analysis of the venous pulse was first aroused in 1927 when, in association with Dr. Cope in the physiological laboratory of the Nebraska Medical School, an attempt was made to determine the duration of the isometric relaxation phase of the cardiac cycle by the use of optical polygraphic tracings.

The apparatus used was a modification of that described by Wiggers (64-65-66). Three Frank segment capsules (12) mounted on miniature cannon carriages, vertically aligned, with chord side out, were used as the recording tambours; thus permitting the use of a photokymograph in which the bromide paper moved vertically. The optional system consisted of an electric bulb, the broad filament of which was focused by convex lenses on the photokymograph after having been reflected by the mirrors of the segment capsules. Displacement due to parallax was obviated and precise time relations were established after the methods suggested by Wiggers,

Dean, Katz and Baker (29-62-63-64-65-66).

Simultaneous records of the heart sounds, venous pulse and carotid were taken on medical students and women dispensary patients. The receiving tambours used were a large stethoscope bell at the apex for sounds, a small circular open bell at the jugular bulb or over the subclavian vein for the venous pulse, and the regulation round flat carotid receiving tambour over the sterno-cleido-mastoid for the carotid pulse. Condom rubber was used over the closed segment capsules recording the carotid and venous pulse. A film of rubber cement was used on the capsule recording the heart sounds, and an open side arm leveled out the slower vibrations of the apex beat (66). All of the subjects used were apparently free from cardiac disease.

Records were obtained which demonstrated a wide variation both in the contour and time relations of the various waves of the venous pulse. In fact it would be possible to select tracings from those we recorded which would lend evidence to almost any of the previously discussed theories as to the mechanism or time relations of the various waves. The explanation for these variations has been discussed previously in this paper. An additional factor may be the following. There can be little doubt, if three or four factors are instrumental in the production of a wave, that they do not maintain an exact ratio to one another at all times in the same individual

or in different individuals. A shift in the ratio of the predominating factors may thus produce a different wave contour or different time relations with respect to other events of the cardiac cycle.

It must be appreciated that each separate venous tracing presents an entirely new problem and must be interpreted as such. Each factor that can be instrumental in the production of each wave must be evaluated. And the conclusion should not be at variance with dynamic possibilities. However, it is probably true that there is a standard mechanism which operates in the majority of normal hearts. And this mechanism can best be appreciated by a consideration of the average time relation of the various waves in a group of records large enough to decrease the influence of the infrequent variations.

The diagram at the close of this paper shows the average time relations of the dicrotic notch, iV, and second sound in a group of technically good records taken from forty-one medical students. All of the records used to illustrate this paper were taken from healthy medical students.

The incisura of the V wave is illustrated in Figures 2, 3, 4, 5, 6, 7, 8 and 15. This incisura apparently has divided the V wave into two definite parts in Figures 3, 5, 6 and 7. In Figures 4 and 8 the incisura is at the foot of the wave. It will be noted that this iV, while variable with respect to the different parts of

the V wave, bears a constant time relationship to the diastolic notch and second heart sound.

The fact that the iV may vary with respect to the V wave in the same individual as well as in different individuals is well illustrated in Fig. 7. In this record the vibrations occurred synchronously with the notch that divided the V wave into two portions in all save the 2nd and 6th cycles. This curve demonstrates that the notch which divides the V wave into two parts is not necessarily caused by the same factor that produces the vibrations. Evidently, at least two separately acting forces operated to produce the V wave in this curve. The first force produced a small primary wave and in its downstroke a second force or group of forces acted to produce a larger wave. The series of vibrations occurred often but not invariably during the interval. Undoubtedly the vibrations are due to the same factor that cause the 2nd sound i.e. the closing of the semilunars. (See page 16)

The iV, when it is a sharp notch, is due to the same cause which produced the vibrations, but it may be complicated by an interval between the forces acting to produce the V wave.

The question naturally arises as to how the iV is transmitted to the jugular bulb where it is recorded. Modes to be considered are - by a pulse wave through the auricle and veins; by sound waves through the auricle and

veins; or by pulse waves or sound waves traveling through an underlying artery. Time relations from selected records might be used to lend support to any of the four above mentioned methods. However, in the majority of cases, as the average time relations illustrated demonstrate, the iV travels as a pulse wave in the underlying artery. The dicrotic notch of the carotid appears just enough later to allow for arterial pulse wave transmission time between the two receiving tambours.

As to the factors which are instrumental in the production of the V wave - the various possibilities have been reviewed previously in this paper. A surprisingly large number of our curves showed the apex of the V wave synchronous with the apex of the dicrotic wave; and it would appear that Gerhardt was as correct in his conclusion that the V wave is transmitted from the artery, as was Mackenzie in his arterial hypothesis for the C wave.

It is very apparent in Figure 8 that the double apex of the V wave is associated with the vibrations of the incisura. Small spikes appear on the V wave of cycles 2 and 3 in Figure 7 due to the same cause. These curves, therefore, might be interpreted in harmony with Barry's hypothesis as to the mechanism of the production of the V wave (3). The fact that there may be a subsequent round shoulder before the onset of Y is illustrated in cycle 8 of Figure 5.

That there is still disagreement as to the mode of the production of the C wave is evidenced by Wiggers' disagreement with other modern investigators. In this regard Figure 16 is very interesting, presenting as it does a technically good record on a healthy student in which there is no demonstrable C wave. It would probably have been prized by Dr. Mackenzie as evidence in an optical record for his theory that there is no C wave in the auricular venous pulse unless from arterial impact. However, the records with a C wave outnumber this one by a hundred to one ratio.

Diastolic waves occurred very frequently in our records and are typical in Figures 1, 2, 3, 5 and 6. The time relations of these diastolic waves as recorded by us are not in accord with the generally accepted idea that they occur synchronously with the third heart sound (11-18-26-53). The position of this third sound may be noted in Figures 12 and 14 from records taken on the writer. There is also an indication of it in cycle 5 of Figure 1. The relative location of the diastolic waves in Figures 1 and 14 would somewhat stretch the transmission time alibi.

Our curves demonstrated a rather frequent occurrence of more than one diastolic wave. Figures 2, 3, 9 and 10. Figures 9 and 10 would support the suggestion of a flip-flop repeating mechanism of Wiggers and Niles for the production of the diastolic waves (68). On superficial examination, the diastolic waves in Figure 10 might

easily be mistaken for the A waves of a fibrillating auricle. The fairly characteristic contour of the venous pulse in the 9th cycle in Figure 9 is a part of the same record.

It is often useful for both clinical and experimental purposes to obtain pulse records with simultaneous pneumographic records. Figure 15 shows such a record.

The technical difficulties in obtaining records are illustrated in practically every curve, especially in the recording of the sounds. In some cases the defect is obvious as in Figure 14 where the venous receiving tambour was in poor apposition and allowed parts or all of the waves to drop out. If in Figure 15 the subject had been suspected of pathology, the diphasic C wave would probably have been regarded as significant. But in all probability it, too, is only indicative of improper application of the receiving tambour.

Many investigators have warned of arterial contamination in the venous pulse, but very few have recognized venous elements in arterial records. Figures 3 and 5 present unquestionable evidence of at least contamination of the arterial tracing by the A wave. Figure 13 shows an arterial (?) tracing which shows contamination by all three venous waves. At the first glance this record appears unintelligible, but practically every detail except the sounds which are obviously artefact, can be explained by physiological events. However, there is no doubt but that it is technically a very poor record.

CLINICAL APPLICATIONS

There is no doubt but that the venous pulse, taken synchronously with the arterial pulse or heart sounds is a good clinical index of intra-cardiac events. In fact, the venous pulse is the only clinical guide to intra auricular pressure changes. For this reason there have been attempts to use the venous pulse in the identification of certain valve lesions, especially tricuspid regurgitation.

Some of the older investigators considered the mere presence of a venous pulse as pathognomonic of tricuspid incompetence. More recently the classification of the venous pulse as auricular and ventricular came into vogue (36). In the later classification was recognized but one atypical form in which there was an absence of the A wave and a fusion of the C and V waves.

Vaquez states that contrary to the opinion of the older authors, examination of the venous circulation gives no indication whatever of the presence or absence of tricuspid insufficiency. He states that the ventricular form does not exist in an organic tricuspid insufficiency, but is often present in tricuspid insufficiency secondary to other cardiac disorders, and especially with auricular fibrillation (55). The usual cause of the ventricular form is obvious. When the auricle is not emptied by the preceding auricular systole, as in auricular

fibrillation, or if there is a stasis or an increase in venous pressure for any other reason, the auricle will fill early and the V wave will anticipate its usual time of production sometimes to the point of completely obliterating the normal systolic collapse. Simultaneous contraction of the auricle and ventricle will also produce this form.

The older investigators were at least partly right as it is very evident that the normal elevations and depressions in the venous pulse are much more apparent in certain pathological states associated with an increase in the venous pressure (24-31-42-58). In this connection it has been found that the venous pulse waves are more prominent during the expiratory phase which is associated with an increase in intra-thoracic pressure (57).

Weber believes that the venous pulse is a pathogenic phenomenon, and is an early symptom of incipient stasis. The premature end of the systolic collapse is, he believes, a sign of beginning circulatory derangement. He states that this symptom occurs long before other methods give any information in regard to the tendency of stagnation (58). Kerr and Warren consider a prominent venous pulse as carrying a grave prognosis (31). However, Straub, without denying the possibility of the occurrence of these signs in venous stasis, maintains that the venous pulse with these variations may occur under physiological conditions (52); and Mackenzie states that the venous

pulsations may be much less prominent in a decompensating individual than they were previously (40).

The venous pulse may also be used in the diagnosis of the various arrhythmias; and has been useful in the past in increasing our knowledge as to the significance of the electrocardiogram (27-35-36-40-49-55-64-65).

When considered in connection with the carotid tracing it has been used to estimate the conduction time from the auricle to the ventricle. A prolongation of the A-C interval over .2 seconds is significant of heart block, and a diminished time indicates ectopic origin of the impulse (55). Wiggers points out the fact that the A-C interval in reality embraces: the systole of the auricle; the intersystolic interval; the period of rising tension in the ventricle; and the transmission time of the arterial pulse in the neck. He regards as "extremely remarkable" the fact that the A-C interval does correspond so well with the electrocardiogram P-R interval (64).

Sinus arrhythmias may be recognized by the variations in the length of the individual cycles in which there is a normal and regular sequence of the A, C, V waves.

Premature contractions when of ventricular origin show no A wave preceding the C-V complex. They generally fall outside of the regular sequence, and may not be associated with arterial waves because of a weak systole. When the ectopic is of auricular origin, the

A-C interval is abnormal, but all three waves appear in the venous tracing. The auricular extra systole may immediately follow a normal systole, but the A wave is always present unless fused with the preceding V.

Auricular fibrillation is characterized by the irregular rapid occurrence of the ventricular C-V complex, with an entire absence of A waves save for small irregular rapid undulations attributed to fibrillation.

Complete heart block shows a regular succession of A waves, interrupted at irregular intervals by C-V complexes, which have a distinctly separate rhythm and thus are occasionally superimposed.

Incomplete heart block is characterized by the presence of a long or progressively increasing A-C interval, which is followed occasionally or regularly by a dropped C-V complex.

In paroxysmal tachycardia the A-C-V sequence is disturbed only by the fact that the V wave may become merged with the succeeding auricular contraction. The record of the onset or termination of the tachycardia is diagnostic.

In auricular flutter when the ventricular rate is relatively slow, there may be a rapid succession of small A waves which are regularly interrupted by the C-V waves.

"It is not always easy to appreciate the various events of the jugular pulse during abnormal action of the

heart" (40). As previously is stated, the technical difficulties in obtaining good records are many.

Practically everything that can be learned from a polygraph can be demonstrated in an electrocardiogram which can be recorded much more easily and quickly; and with a better chance of the record being intelligible.

The interest in graphic methods grew out of a desire to observe the pathological alterations of the different properties of the heart tissue i.e. rhythmicity, excitability, stimulus production, conductivity, tonicity and contractility. The polygraph can demonstrate the last named property as in pulsus alternans, while the electrocardiogram is silent concerning this irregularity in volume. However, the electrocardiogram is more exact in the recording of the other demonstrable properties of heart muscle.

The venous pulse because it is a volume curve still has a large part to play in the understanding of the pathological and physiological dynamics of the heart. It is, of course, valueless unless there is a synchronous record of the sounds or arterial pulse. The registration of the sounds as demonstrated in the curves of this paper should be particularly useful in observing the variation of heart sounds during illness, and in rendering the appraisal of murmurs less a matter of opinion and more a matter of scientific record.

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CONCLUSIONS

The proper analysis of a technically good venous tracing, recorded simultaneously with carotid pulsations and the heart sounds, gives useful and extensive information concerning the successive normal and pathological dynamic events in the heart.

Such a record is useful in determining the duration of the different phases of the cardiac cycle, in demonstrating the abnormal volume changes associated with certain valve lesions, in deciphering the mechanism of the arrhythmias, and in furnishing permanent graphic records; thus rendering the appraisal of clinical signs less a matter of opinion and more a matter of scientific record.

The registration of an intelligible venous pulse is accompanied by many more technical difficulties than is the registration of an electrocardiogram; and for this reason the latter is the method of choice in routine clinical work.

A great deal of variation still exists among accurate observers as to the interpretation of the venous pulse both in health and disease. Because of this, a wide field is open to the skilled observer in the determination of the time relations, and of the various forces instrumental in changing the contour of the venous pulse.

In the publication of work concerning the venous

pulse, the results, i.e. the records and time relations as well as the conclusions drawn from the results, should have an important part; and the standard mechanism which operates in the majority of cases should be determined by the consideration of a group of selected records large enough to decrease the influence of the infrequent variation.

In the interpretation of venous tracings each record should be considered as a separate problem. Every factor that can be instrumental in the production of a wave should be considered; and the conclusions must not be at variance with dynamic possibilities.

The writer's results indicate that the V wave is produced by at least two separately acting forces; and that the incisura of the V wave usually reaches the jugular receiving tambour via an arterial pulse wave.

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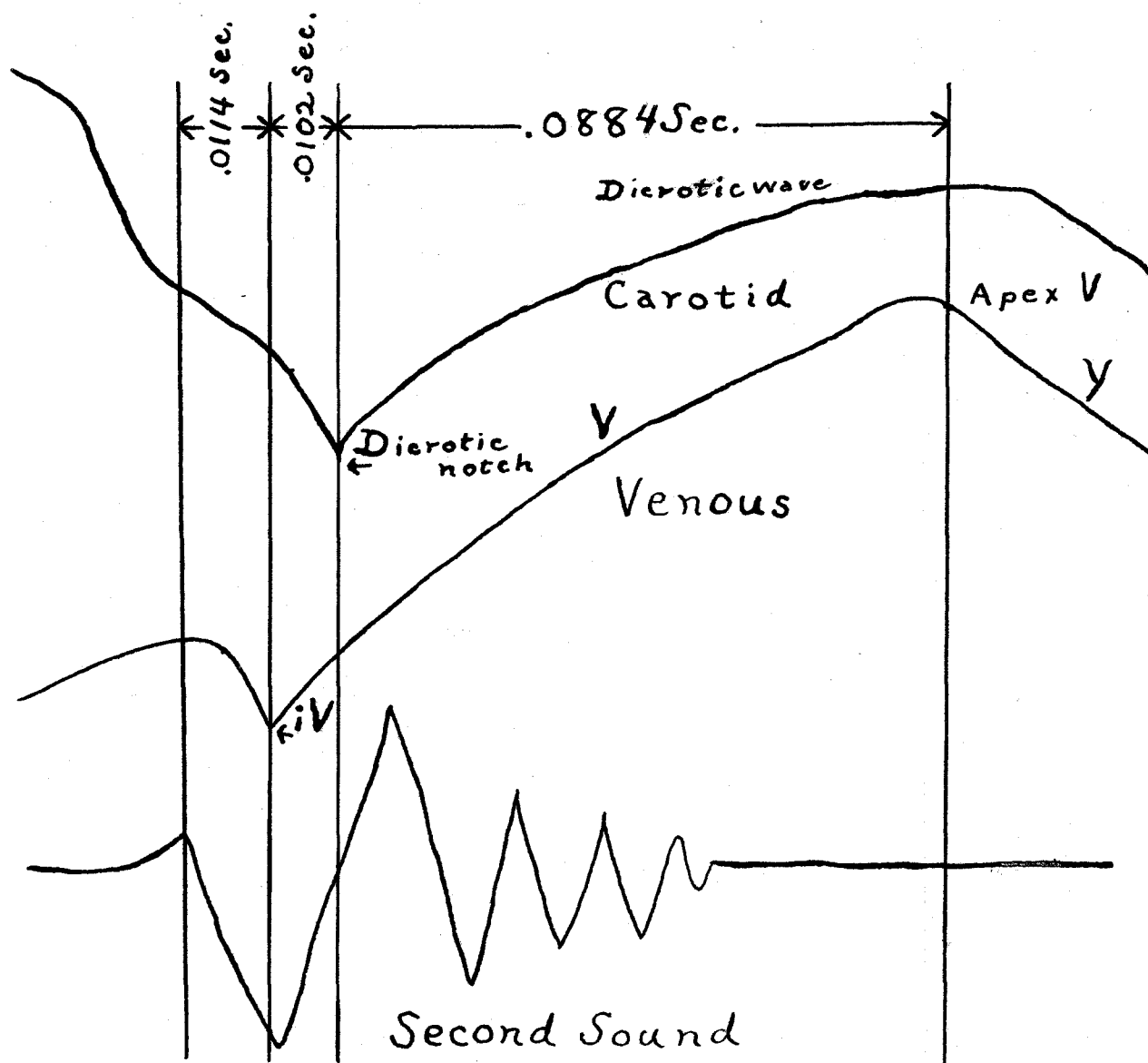


Diagram of the average time relations of the dicrotic notch, incisura of the V wave, and onset of the second sound from records taken on forty-one healthy medical students.

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